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Genetic control of photorespiration in plants

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Abstract

Photorespiration is a pervasive process in C_3 plants, initiated by the oxygenation reaction of Rubisco, which yields 2-phosphoglycolate, a toxic metabolite that inhibits the Calvin Cycle. To mitigate its toxic effects and recover carbon, plants have evolved a multi-organelle photorespiratory pathway involving chloroplasts, peroxisomes, mitochondria, and cytosol. Though traditionally labeled as an energetically wasteful process, regulation of this pathway is of great importance under current and future environmental changes, including rising temperatures, atmospheric CO_2 levels, and varying light intensity, due to trade-offs among carbon assimilation, energy expenditure, redox balance, and stress tolerance. Genetic control of this pathway lies at the core of photorespiratory regulation, with genes encoding enzymes, metabolite transporters, regulatory proteins, transcription factors, and even post-translational modifications (PTMs) modulating flux. In this review, we synthesize our current knowledge of gene-level control of photorespiration in plants. Key themes include canonical enzyme genes, viz., glycolate oxidase, glycine decarboxylase, hydroxy pyruvate reductase, aminotransferases, genetic variants and mutants, transcriptional regulation under environmental cues, non-canonical/alternative pathways, organ/cell-type specificity, and emerging regulatory layers such as PTMs and epigenetics. This review also highlights critical knowledge gaps and suggests future directions in genetic engineering and meta-analytic quantification to enhance photosynthetic efficiency, stress tolerance, and climate resilience in plants.

Keywords: Photorespiration, Rubisco oxygenation, canonical enzymes, transcription factors, post-translational modifications, photosynthetic efficiency

1. Introduction

Photorespiration has long been considered a wasteful side activity of Rubisco's oxygenase reaction. However, over the past decades, studies have overturned this simplistic view by revealing its importance as an essential repair mechanism, its protective role against photodamage and reactive oxygen species (ROS), and its integration with nitrogen and redox metabolism [1]. As global temperatures rise and atmospheric CO_2 concentrations fluctuate, the relative rate of Rubisco oxygenation tends to increase, raising the cost of photorespiration [2]. Genetic control of the photorespiratory pathway thus becomes central, and it's important to know which genes are active, how they are regulated, and how genetic variation can either mitigate or exacerbate the costs associated with photorespiration.

In this review, we aim to collate what is known about the genetic control of photorespiration in plants, including the enzyme-coding genes, their regulation, mutants, natural variation, alternative pathways, and regulatory networks. Here, our goal is to present a cohesive picture that highlights what interventions (breeding, engineering) might be most effective and where data are lacking, especially the challenges posed by climate change.

2. Overview of the photorespiratory pathway

Before diving into genetic control, it is important to review the photorespiratory pathway. The photorespiratory pathway begins when Rubisco acts on O_2 instead of CO_2 . RuBP (ribulose-1, 5-bisphosphate) is oxygenated, yielding one molecule 3-phosphoglycerate (3-PGA) usable in the Calvin cycle, and one molecule 2-phosphoglycolate (2-PG), which is an inhibitory metabolite to key enzymes in photosynthesis (e.g., Triosephosphate isomerase, sedoheptulose-1, 7-bisphosphatase) [3,4]. Plants detoxify 2-PG via a series of steps (Fig. 1)

- **Chloroplast:** phospho glycolate phosphatase (PGLP) removes phosphate from 2-phospho glycolate → glycolate.
- **Peroxisome:** glycolate oxidase (GOX) oxidizes glycolate → glyoxylate + H₂O₂; aminotransferases (GGAT/SGAT) convert glyoxylate to glycine. Catalase removes H₂O₂.
- **Mitochondrion:** Glycine decarboxylase complex (GDC) converts glycine → serine, releasing CO₂ and NH₃; serine hydroxymethyl transferase (SHMT) often works in tandem.
- **Back in peroxisome/cytosol/chloroplast:** hydroxy pyruvate reductase (HPR), glycerate kinase (GLYK), and transporters shuttle intermediates back into the Calvin cycle.

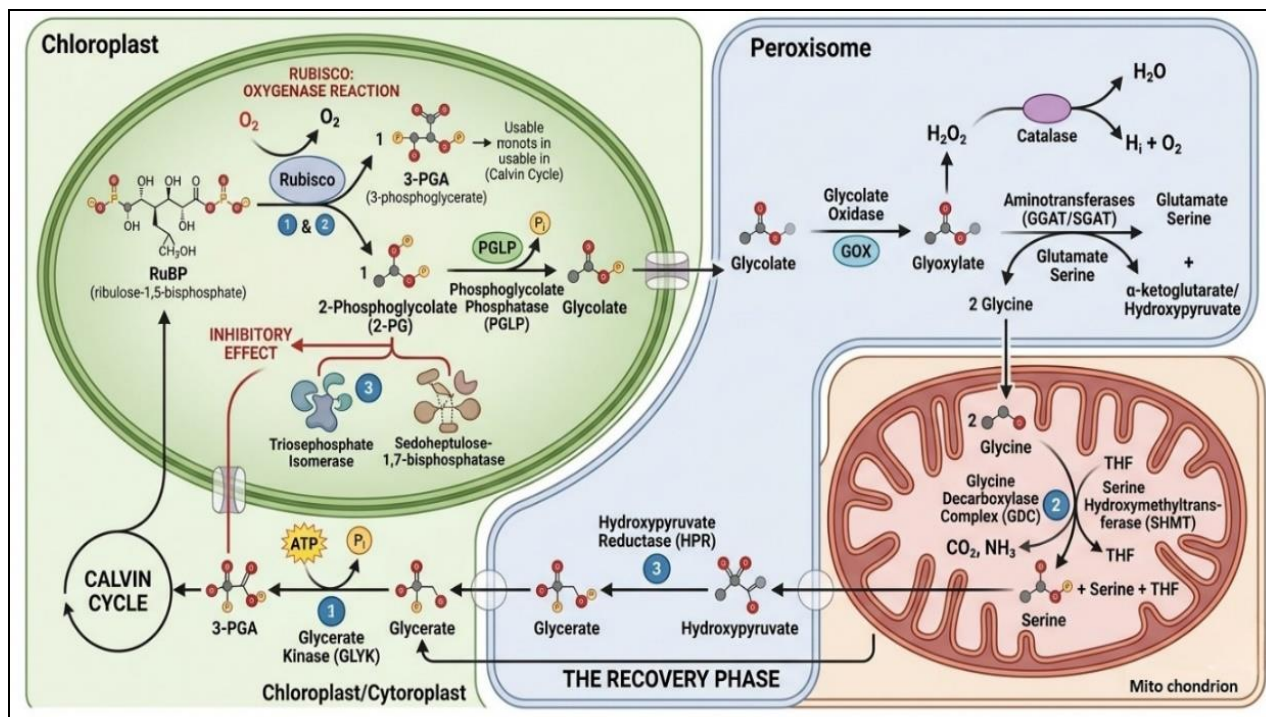


Fig 1: Overview of the photorespiratory pathway

Understanding the genes encoding all these enzymes, their isoforms, regulatory kinetics, and expression under various conditions is essential to mapping genetic control. Table 1 summarizes the major photorespiratory genes, the enzymes they encode, associated mutant phenotypes, and their functional roles in both canonical and alternative photorespiratory processes.

3. Canonical enzyme genes and their mutants

This section reviews the major enzymes involved in photorespiration, with emphasis on evidence derived from genetic mutant and functional characterization studies.

3.1 Phospho glycolate phosphatase (PGLP)

PGLP catalyzes the first committed step in the detoxification of 2-phosphoglycolate. Genetic studies, particularly in C₃ species, reveal that mutations in PGLP severely compromise normal growth under ambient conditions. Although there are fewer isoforms of PGLP present compared to other enzymes, their expression patterns and allelic variation are crucial for flux control, particularly under low CO₂ or high light stress. [5, 6]

3.2 Glycolate oxidase (GOX) and Catalase

Glycolate oxidase (GOX) has multiple isoforms (GOX1 and GOX2) in *Arabidopsis*. Loss of GOX1, but not GOX2, alleviates the photorespiratory stress phenotype of catalase-deficient *cat2-2* mutants, indicating that GOX1 plays a more prominent role under specific photorespiratory flux conditions [7, 8]. Mutations in GOX result in altered

metabolite accumulation at low CO₂ levels. For instance, *gox* mutants in *Arabidopsis* under low CO₂ treatments have disturbed Gly/Ser levels and broad reconfiguration of other amino acid pools [9]

Catalase (CAT) functions closely with GOX by detoxifying the H₂O₂ generated during glycolate oxidation. In *Rhazya stricta*, increased activities of PGP and CAT accompany greater photorespiratory flux at high temperatures, suggesting genetic upregulation of CAT is an adaptive trait [2]

3.3 Glycine decarboxylase complex (GDC)

The **Glycine decarboxylase complex (GDC)** comprises multiple subunits of H, T, P, and L proteins that are localized in mitochondria and function as a central component of the photorespiratory pathway. Mutants of GDC components often show severe phenotypes under ambient conditions (low CO₂), because glycine accumulates and downstream reactions stall. For example, *Arabidopsis* mutants deficient in glycine decarboxylase show reduced photosynthetic rate and growth unless CO₂ is elevated [10] also more recent guard-cell specific manipulations, In a recent study the GDC-H (H-protein) in guard cells (GC) in *Arabidopsis* were manipulated and when guard cell GDC-H expression was increased, the plants showed improved growth, photosynthesis, carbohydrate accumulation, and better stomatal behavior under high photorespiratory demand, showing that GDC in guard cells plays important regulatory roles in guard cells beyond its conventional role in mesophyll enzyme function [11]

3.4 Aminotransferases: GGAT/SGAT/Serine-glyoxylate aminotransferase

In *Arabidopsis thaliana*, aminotransferases such as GGAT and SGAT are essential for photorespiratory amino acid interconversions. Aminotransferases convert glyoxylate to glycine (GGAT & SGAT) or catalyze serine/glyoxylate interconversions. Mutants lacking serine-glyoxylate aminotransferase activity in *Arabidopsis* are lethal under ambient photorespiratory conditions. Under conditions that promote photorespiration, these mutants accumulate glycine and serine, with greatly reduced photosynthesis and starch and sucrose contents. However, the mutants can survive under conditions that suppress photorespiration, highlighting the essential role of SGAT in maintaining photorespiratory metabolism and carbon balance [12]

Gene variants in glutamate: glyoxylate aminotransferase (GGAT) are also implicated in ozone sensitivity and oxidative stress responses, meaning that these aminotransferases are part of the interface between photorespiration and environmental stress. [13] This finding emphasizes the broader physiological role of photorespiratory enzymes in stress adaptation and redox homeostasis

3.5 Hydroxy pyruvate reductase (HPR)

Hydroxypyruvate reductase (HPR) catalyzes the conversion of hydroxypyruvate to glycerate, an essential step in the photorespiratory pathway. In *Arabidopsis*, *hpr1* mutants exhibit phenotypes consistent with impaired photorespiration, including accumulation of hydroxypyruvate and reduced growth/photosynthesis under ambient or high photorespiratory flux [14]. Also, recent identification of suppressor mutants reveals “non-canonical” pathways that can partially compensate for the loss of HPR1 function. In particular, cytosolic glyoxylate reduction mediated by HPR2 has been suggested to provide a bypass mechanism that supports continued photorespiratory metabolism under conditions of HPR1 deficiency [15].

4. Alternative Pathways and Genetic Modifiers

In addition to the classical canonical pathway, recent genetic research has uncovered modifiers and non-canonical routes. These non-canonical pathways can partially compensate for defects in core photorespiratory enzymes, thereby

improving metabolic flexibility under stress or mutant conditions. Genetic analyses of suppressor mutants have identified bypass reactions involving cytosolic and peroxisomal enzymes, indicating that plants have backup strategies to preserve redox balance and carbon recycling in the event that the principal pathway is disturbed. These results show that the pathway functions as a part of a larger, interconnected metabolic network rather than as a purely linear process, highlighting the intricacy of photorespiratory control.

4.1 Glyoxylate Shunts and Non-canonical Routes

The recently described **cytosolic glyoxylate shunt** in *Arabidopsis* complements the canonical photorespiratory pathway when HPR1 (hydroxy pyruvate reductase) is deficient. The gene GLYR1 (glyoxylate reductase 1) helps convert excess glyoxylate to glycolate in the cytosol, which then, through HPR2, re-enters the canonical cycle. Suppression of *hpr1* by *gyr1* mutants partially rescues growth under high photorespiratory flux or deficiency of canonical pathway components [15]. These findings suggest that plants possess metabolic flexibility and redundancy, which are useful under stress or when canonical genes are impaired.

4.2 Transporter Genes and Subcellular Localization

Transport across organelles is essential for the photorespiratory cycle: intermediates like glycolate, glyoxylate, glycerate must move between the chloroplast, peroxisome, mitochondrion, and cytosol. Genetic variation in transporter genes (e.g. PLGG1, BASS6, various plastid envelope transporters) can strongly affect flux, though the number of well-characterized transporter mutants is still fewer than for enzyme genes.

Subcellular localization (where enzyme isoforms are expressed) also influences genetic control: for example, GOX1 vs GOX2 (peroxisomal GOX isoforms) in *Arabidopsis* show functional divergence: GOX1 plays a more critical role under photorespiratory stress, whereas GOX2 plays a less critical role. [7]. These findings suggest that spatial distribution and differential expression of enzyme isoforms provide an additional layer of metabolic specialization and regulatory control within the photorespiratory network

Table 1: List of genes or gene family associated with Photorespiration and Related Metabolism

Gene/Gene Family	Primary Function in Photorespiration and Related Metabolism
RBCS/RBCL (Rubisco small and large subunits)	Catalyzes the carboxylation and oxygenation of RuBP; initiates both photosynthesis and photorespiration. Altered expression affects photorespiratory flux and CO ₂ /O ₂ specificity [16]
PGLP (2-Phosphoglycolate Phosphatase)	Dephosphorylates 2-phosphoglycolate to glycolate in the chloroplast; first enzymatic step of the photorespiratory pathway [5].
GOX (Glycolate Oxidase)	Converts glycolate to glyoxylate in the peroxisome, generating H ₂ O ₂ ; central to glyoxylate cycle regulation [6]
CAT (Catalase)	Detoxifies H ₂ O ₂ produced by GOX; maintains peroxisomal redox homeostasis during photorespiration [9]
GGAT (Glutamate: Glyoxylate Aminotransferase)	Transfers amino groups to form glycine from glyoxylate; important for linking nitrogen and carbon metabolism [17]
GDC Complex (P-, H-, T-, and L-proteins)	Mitochondrial glycine decarboxylation system; releases CO ₂ and NH ₃ while producing serine precursor; controls photorespiratory CO ₂ loss [18]
SHM (Serine Hydroxy methyl transferase)	Converts glycine to serine in mitochondria; produces 5, 10-methylenetetrahydrofolate for one-carbon metabolism [12, 19]
HPR (Hydroxy Pyruvate Reductase)	Reduces hydroxy pyruvate to glycerate in peroxisomes; essential for recycling 3-phosphoglycerate into Calvin cycle [20]
GLYK (Glycerate Kinase)	Phosphorylates glycerate to 3-PGA in chloroplasts; final enzymatic step closing the

	photorespiratory cycle ^[21, 22]
PLGG1 (Plastidic Glycolate/Glycerate Transporter 1)	Transports glycolate and glycerate between chloroplast and peroxisome; crucial for photorespiratory metabolite exchange ^[23, 24]
BASS6 (Bile Acid Sodium Symporter 6)	Transports glycolate across chloroplast envelope; complements PLGG1 function ^[25]
PDP (Peroxisomal Dicarboxylate Transporter)	Exchanges organic acids (malate, 2-oxoglutarate) influencing peroxisomal redox status during photorespiration ^[26, 27]
SGAT (Serine:Glyoxylate Aminotransferase)	Converts serine + glyoxylate → hydroxy pyruvate + glycine; coordinates glycine/serine interconversion and nitrogen assimilation ^[17]
PGLP1/PGLP2 Isoforms	Differentially expressed in chloroplasts; PGLP1 predominant under high photorespiratory flux ^[28]
HPR1/HPR2 Isoforms	HPR1 is peroxisomal NADH-dependent; HPR2 cytosolic NADPH-dependent, providing redundancy under stress ^[20]
PGRP (Photorespiratory Glycolate Recycling Proteins)	Novel group of glycolate-processing enzymes enhancing photorespiratory flux; identified via transcriptomics under low CO ₂ ^[17, 23]
Fd-GOGAT/GS2 (Ferredoxin-dependent Glutamate Synthase/Chloroplastic Glutamine Synthetase)	Reassimilate photorespiratory NH ₃ in chloroplast; maintain nitrogen balance and minimize toxicity ^[29]
CAT2/CAT3 Isoforms	Differential catalase isoform activity fine-tunes peroxisomal ROS detoxification under fluctuating light ^[30]
PPDK/PEPC (Secondary Pathway Genes)	In some C ₃ -C ₄ intermediates, these enzymes partially recycle photorespiratory CO ₂ via C ₄ -like metabolism ^[31]
PGLP3/GOX3 Mutants (Arabidopsis)	Mutant studies revealed feedback signaling to nuclear gene expression controlling metabolic compensation ^[32]
ROG1/ROG2 (Regulators of Glyoxylate Metabolism)	Participate in signaling and transcriptional regulation of peroxisomal enzymes ^[17]
RBOH (Respiratory Burst Oxidase Homologs)	Coordinate ROS signaling from peroxisome to chloroplast, modulating photorespiratory gene expression ^[17]
ABI4/HY5/PIF4 (Transcription Factors)	Integrate light and stress signals with photorespiration gene regulation ^[33]
NAD-ME/MDH (Malate Enzyme/Malate Dehydrogenase)	Support redox balance between organelles during photorespiratory flux ^[17]
H ₂ O ₂ -responsive WRKY and NAC TFs	Regulate peroxisome-to-nucleus signaling during high photorespiration stress ^[34]

5. Regulation of Photorespiratory Genes under Environmental and Developmental Signals

Gene activity in photorespiration is not constitutive; many genes respond dynamically to environmental and developmental cues.

5.1 CO₂ Levels and Light Intensity

Low CO₂ conditions promote expression of many photorespiratory genes. Transcriptomic studies in *Arabidopsis* and legumes such as *Lotus japonicus* have demonstrated coordinated regulation of several pathway components, including *GLU1/GLU2*, *PGLP*, and *GOX*, in response to changes in CO₂ availability and light conditions. ^[35] These responses indicate tight integration between photorespiration, carbon metabolism, and environmental signaling pathways. In *Chlamydomonas reinhardtii*, low CO₂ shifts induce expression of the Calvin cycle, CCM (carbon concentrating mechanism), and photorespiratory genes, but some with transient dynamics, showing that gene induction timing matters ^[36]

Light intensity/high light often increases expression of *GOX*, *CAT*, *PGP* and related genes, as well as prompting increased enzyme activity (e.g. in *R. stricta*, *Arabidopsis*) to handle the increased oxygenation burden under high light ^[2]

5.2 Temperature, stress, and redox Signals

Elevated temperature increases the rate of Rubisco oxygenation, thereby increasing flux through the photorespiratory pathway. In the extremophile *Rhazya stricta*, plants maintain high photorespiratory enzyme levels (*PGP*, *CAT*) and conductances that have been reported to support efficient carbon metabolism and reduce metabolic limitations under high-temperature conditions ^[2].

Stressors such as ozone, oxidative stress, and even fluctuations in O₂ partial pressure (or inhibitors) influence

gene expression and/or activity. For example, mutants of *GOX* and *GAT* show enhanced sensitivity under ozone, indicating both enzyme abundance and regulation matter for stress resilience ^[13].

Low CO₂ conditions impose carbon-limited stress (carbon starvation), triggering metabolic and transcriptional adjustments in plants. Many transcriptomic studies have revealed upregulation of certain photorespiratory genes under such conditions, along with the accumulation of photorespiratory intermediates, including glycine and serine, as well as branched-chain amino acids ^[37]. These responses reflect the close association between photorespiration, carbon metabolism, and stress adaptation pathways.

5.3 Developmental stage and cell-type specificity

Recent work shows that photorespiration genes are not only modulated by temporal cues but also by spatial and cell-type-specific mechanisms. In particular, differential regulation between mesophyll and guard cells has emerged as an important component of photorespiratory control. The guard cell (GC) specific manipulation of *GDC-H* in *Arabidopsis* demonstrates that GC photorespiration influences stomatal behavior, growth, and carbohydrate metabolism. Varying GC *GDC-H* expression correlates with differences in stomatal conductance and photosynthetic induction under photorespiratory demand ^[11]. In addition, the developmental stage appears to affect photorespiratory responsiveness, as young leaves vs mature leaves often exhibit different capacities for gene induction under stress, though few systematic studies have compared leaf age or developmental stage across diverse species.

6. Regulatory Layers Beyond Gene Expression

Regulation of photorespiration extends beyond changes in gene transcription and involves several additional levels of

control. In addition to simple transcriptional control, genetic/epigenetic regulatory layers modulate photorespiration. Together, these processes help plants rapidly adjust their photorespiratory activity in response to environmental and developmental conditions.

6.1 Transcription Factors

While many enzyme genes are known, fewer transcription factors (TFs) that specifically regulate photorespiratory genes are well characterized. Some TFs that respond to light, CO₂, sugar status, redox are candidate regulators. For example, in *Lotus japonicus*, the lack of plastidic glutamine synthetase led to the coordinated repression of multiple photorespiratory genes, suggesting higher-level regulation, possibly via sugar-or nitrogen-signaling TFs [38]

In *Chlamydomonas*, the transcription factor CIA5 is required for the induction of many Carbon Concentrating Mechanism (CCM) genes and also influences the expression of photorespiratory genes during shifts from high to low CO₂ [36]

6.2 Post-translational Modifications (PTMs)

Gene expression alone doesn't determine enzyme activity: PTMs, including phosphorylation, redox modulation, and possibly ubiquitination, are emerging as important control points.

For example, increased activity of GOX and CAT under heat stress suggests not just upregulation of transcripts but perhaps stabilization, activation, or reduced turnover of those proteins [2]. Though detailed data on specific PTMs in many photorespiratory enzymes remain sparse, there is evidence in the literature (outside the most recent works) that redox state and ROS can modulate enzyme activity directly.

6.3 Epigenetic Control

Epigenetic regulation, including DNA methylation and histone modifications, might modulate how photorespiratory genes respond to environmental and developmental factors. To date, direct evidence for epigenetic regulation of core photorespiratory pathway genes is limited, but transcriptomic analyses conducted in mutant backgrounds suggest the possible involvement of chromatin-level regulatory processes. These observations suggest that epigenetic regulation may influence the responsiveness and coordination of photorespiratory metabolism, though further investigation is needed to clarify the underlying mechanisms [39].

7. Genetic Engineering Approaches and Crop Implications

Given the importance of photorespiration in cost and yield under stress, there has been substantial interest in engineering plants to improve the photorespiratory pathway through genetic and physiological approaches. Here is a breakdown of how researchers are manipulating photorespiration to boost crop yields and resilience.

- **Overexpression of key enzymes:** Some studies show that overexpressing or increasing the expression (via transgenics) of enzymes like PGP, CAT, or GOX can increase photosynthetic capacity and growth under high light or heat (e.g., in *R. stricta*, *Arabidopsis*) [8, 40, 41]

- **Guard cell targeted manipulation:** The recent GC-specific GDC-H work suggests that manipulating photorespiration in epidermal/guard cell tissues can influence stomatal behavior, thereby indirectly influencing CO₂ uptake and water-use efficiency [11]
- **Natural variation and selection:** Some species (extremophiles) have naturally elevated levels or activities of photorespiratory enzymes, or favorable conductances, enabling them to tolerate high temperature photorespiratory pressure [2]. These can be utilized as models or donors in breeding.
- **Trade-offs:** Genetic modifications need to account for potential cost in redox balance, ammonium assimilation, ROS management, and growth in benign vs stress conditions. [42]

8. Knowledge Gaps and Future Perspectives

Despite considerable progress, there are several persistent gaps in our understanding of the genetic control of photorespiration:

1. **Lack of effect size quantification:** Many studies report fold-changes, expression, activity, but do not report full statistical details (means, variability, replicates) necessary for comparing across species or for meta-analysis.
2. **Limited crop-scale data:** Most detailed genetic work has been done in *Arabidopsis* and a few model species. Very few extensive studies exist in major crops (rice, wheat, maize) that connect gene manipulations to physiological outcomes under field or near-field conditions.
3. **Temporal and developmental dynamics:** Leaf age, developmental stage, and time-series under fluctuating environmental conditions (light/dark transitions, heat stress onset/relief) are under-studied.
4. **Cellular and tissue specificity:** Aside from recent guard cell work, little is known about how different tissues contribute to whole-plant photorespiratory regulation.
5. **Non-canonical pathways:** The newly discovered shunts (e.g., cytosolic glyoxylate shunt) need further genetic characterization: which species have them, how they are regulated, their capacity, how they interact with the canonical pathway under diverse environments.
6. **Regulatory proteins (TFs, co-factors) and PTMs:** Identification of specific transcription factors that control clusters of photorespiratory genes is incomplete; PTMs (and possibly epigenetic modifications) have been less explored.
7. **Integration with other metabolic and signaling pathways:** Ammonium assimilation, nitrogen metabolism, ROS/redox signaling, and photosynthetic electron transport all feed into or are affected by photorespiration. How genetic variants in photorespiration affect these networks globally is insufficiently known.
8. **Natural variation and adaptive evolution:** There is room for genome-wide association studies (GWAS), QTL mapping in diverse germplasm to identify allelic variation of photorespiratory genes that confer stress tolerance or efficiency.

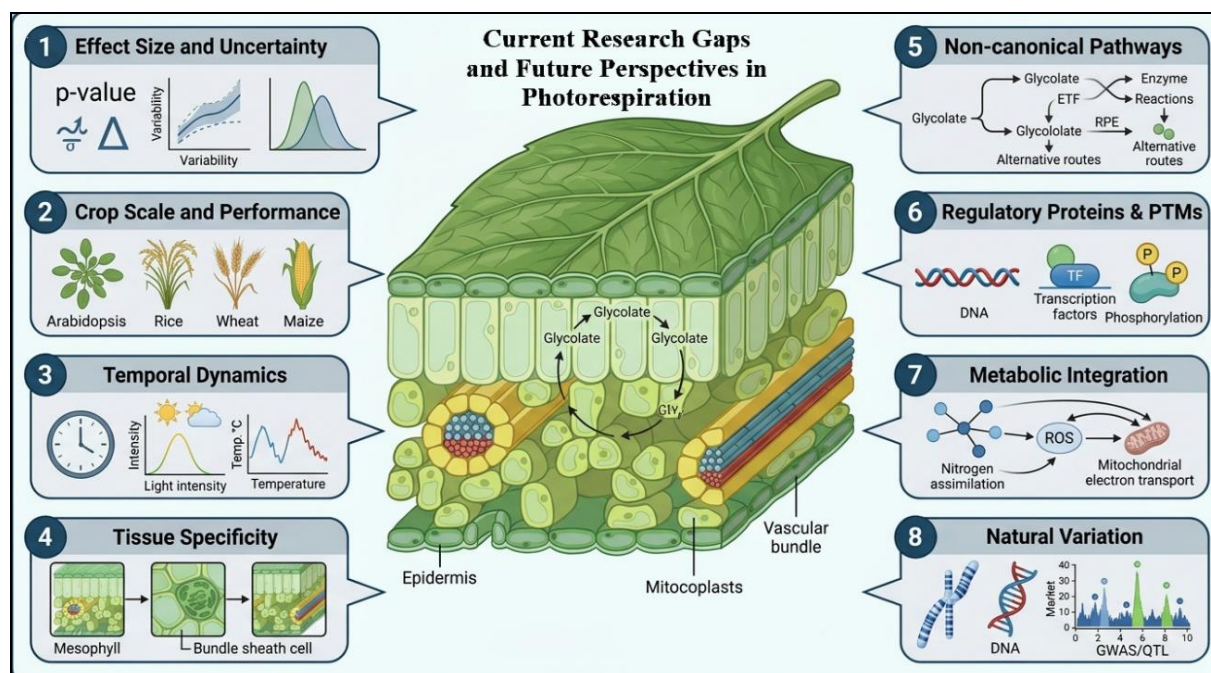


Fig 2: Current Research Gaps and Future Perspectives in Photorespiration

9. Conclusions

Genetic control of photorespiration in plants is multi-layered and dynamic. Core enzyme genes (GOX, GDC, HPR, aminotransferases, PGLP) are well characterized in mutants, showing their essential roles under ambient photorespiratory load. Variation in isoforms (e.g., GOX1 vs GOX2), alternative pathways (e.g., the glyoxylate shunt), and cell-type specificity (e.g., guard cells) now add complexity to the regulatory picture. Environmental signals like CO₂, light, temperature, and stress modulate expression and activity, but many regulatory layers—transcription factors, PTMs, epigenetics—remain incompletely described. For breeding or engineering crops with improved photosynthetic efficiency under climate stress, leveraging natural variation, precise gene manipulation, and detailed analysis of integrated outcomes (photosynthesis, growth, water-use, nitrogen, ROS) will be essential.

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